

Cryptogenic Isolated Cortical Venous Infarct: A Report of Three Cases

Ertuğrul Uzar¹, Atilla İlhan¹, Banu Çakır², Alevtina Ersoy¹, Ülkühan Düzgün¹, Burak Uz³

¹Department of Neurology, Faculty of Medicine, Fatih University, Ankara, Turkey

²Department of Radiology, Faculty of Medicine, Fatih University, Ankara, Turkey

³Department of Internal Medicine, Faculty of Medicine, Fatih University, Ankara, Turkey

ABSTRACT

Cortical vein infarction without dural sinus involvement is extremely rare. Herein, we present three patients with headache, partial seizure and right-sided numbness. On neurological examination, focal neurologic deficit was not observed in our patients. Magnetic resonance imaging revealed cerebral ischemia which showed as hypointense on T1-weighted images and hyperintense on T2-weighted images that do not follow the boundary of arterial territories, indicating cortical venous infarct. Cortical venous infarct should be suspected in patients who present with sudden onset headache and/or focal epileptic seizures even if there is no neurologic deficit. The diagnosis and treatment of cortical venous infarct should be considered as an emergency because of the high potential for full recovery with anticoagulant treatment.

Key Words: Cortical venous infarct, seizure, anticoagulant treatment

Received: 20.12.2009

Accepted: 29.03.2010

Introduction

Nonhemorrhagic cortical infarcts are only seen in 4% of cerebral venous thrombosis (CVT) cases (1). Cortical vein infarction (CVI) without dural sinus involvement has rarely been reported. The spectrum of clinical symptoms depends on the localization of venous infarction. However, CVI is a rare cause in patients who are admitted to the hospital with focal seizures (2, 3). Herein we present 3 cases with CVI in patients without dural sinus thrombosis. These patients were admitted to our hospital with symptoms of progressive headache, partial seizure and right sided numbness. We also discussed the patients' characteristics and identified the factors that may contribute to the diagnosis and prognosis.

Case Reports

Case 1

A 41 year-old male patient presented to our hospital with progressive right frontal sided headache that began 2 weeks earlier. Subsequently, he suffered from a sudden numbness on the right side. However, the neurologic examination and computerized tomography (CT) scan of the brain were unremarkable. Cranial magnetic resonance imaging (MRI) examination was performed. A lesion which was hypointense on T1-weighted images and hyperintense on T2-weighted images was observed in the right temporal lobe and superior temporal gyrus involving the cortex and subcortical white matter (Figure 1A). The lesion did not follow the boundary of classical arterial territories. On diffusion-weighted images, restricted

diffusion was observed in this area. Following intravenous gadolinium administration, parenchymal contrast enhancement especially as a gyral pattern was detected (Figure 1B). MR venography revealed venous vessels within normal limits (Figure 1C). On the basis of MRI findings venous infarction was considered. Lumbar puncture was performed to rule out infection. Cerebrospinal fluid (CSF) examination was normal. A prothrombotic screen including proteins S and C, activated protein C resistance, antithrombin III, factor V Leiden mutation, MTFHR C677T, von Willebrand factor activity, and homocysteine levels were all normal. ANA and Anti-ds-DNA were also negative. Transthoracic echocardiography, cranial MR arteriography and venography and carotid MR angiography findings were unremarkable. He was treated with low molecular weighted heparin (LMWH). After two days, complete resolution of his symptoms was achieved.

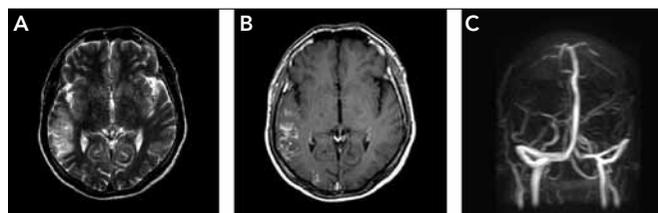


Figure 1. A, B, C. A lesion which is located in the right temporal lobe and superior temporal gyrus involving cortex and subcortical white matter is seen hyperintense on axial T2-weighted image (1A). Postcontrast axial T1-weighted image (1B) shows contrast enhancement especially as a gyral pattern. MR venography shows venous vessels within normal limits (1C)

Case 2

A 57 year-old woman was admitted to our hospital with sudden onset of right-sided numbness that resolved in 5 minutes. Her past history was unremarkable for vascular diseases. The neurologic examination and cranial CT were found to be normal, MRI depicted a lesion in the left postcentral gyrus which was hypointense on T1-weighted images and hyperintense on T2-weighted images. Axial fluid-attenuated inversion recovery (FLAIR) image (Figure 2A) revealed a hyperintense lesion in the left postcentral gyrus. Diffusion weighted MR image (Figure 2B) showed no restriction in this area. On postcontrast T1-weighted images, contrast enhancement was not detected. On the basis of MRI findings venous infarction was considered in the primary differential diagnosis. Therefore, LMWF and acetyl salicylic acid were given to the patient. Prothrombotic and CSF screens were all normal. A follow-up MRI examination was performed after 20 days. Although the dimension of the lesion was the same, the edema area in the anterior part of the lesion was minimally regressed on T2-weighted images. On postcontrast-images minimal contrast enhancement in the anterior part of the lesion was observed. Three weeks after admission to the hospital, the patient had a clonic seizure of her right upper extremity. Therefore valproic acid treatment was administered to the patient for partial epilepsy which might be due to venous infarction. At the follow-up examinations the seizures did not recur.

Case 3

A 43-year-old male patient was admitted to our hospital with a clonic seizure of his left arm. The cranial CT was normal. One week earlier, the patient had a secondary tonic-clonic seizure. The neurologic and systemic examinations were normal. Laboratory studies and EEG were found to be normal. MRI showed an edematous lesion involving the subcortical white matter in the right precentral gyrus (Figure 3A-C). After administration of intravenous gadolinium, minimal linear contrast enhancement in the neighboring cortex was seen. On diffusion-weighted images, increased diffusion was observed in this region. Venous infarction was primarily considered in the differential diagnosis. LMWF and acetyl salicylic acid were given to the patient. In addition, valproic acid 500 mg/day was started for partial seizures and at the follow-up the dose was increased to 1000 mg/day. The seizures were not observed and the patient was discharged. However, after 2 weeks of discharge, the patient had 3 episodes of focal motor seizures. Valproic acid was stopped and phenytoin 300 mg/day was started. Because of the repeated seizures, fenitoin was changed to levetirasetam 1000 mg/day. The seizures recovered completely. An MRI after 1 month demonstrated considerable regression of the hyperintense signal abnormality. On follow up, 8 months after the initial incident, the MRI image showed complete resolution of the pathological signal alteration.

Discussion

Cerebral veins do not have valves, hence the occlusion of the cerebral venous flow by a partially obstructing throm-

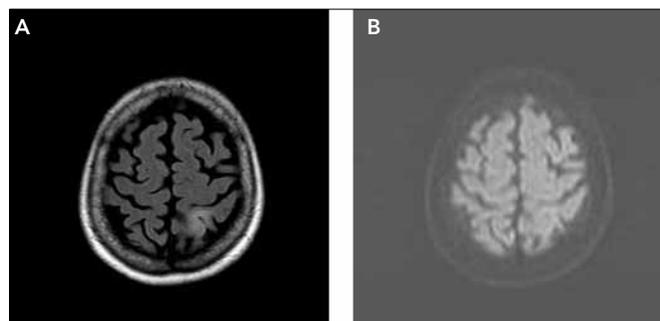


Figure 2. A, B Axial fluid-attenuated inversion recovery (FLAIR) image (2A) shows a hyperintense lesion in the left postcentral gyrus. Diffusion weighted MR image (2B) shows no restriction in this area

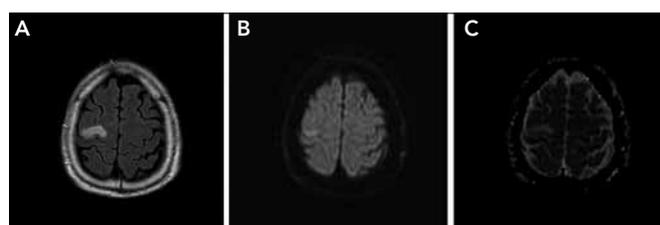


Figure 3. A, B, C. The lesion in the right precentral gyrus is seen as hyperintense on axial fluid-attenuated inversion recovery (FLAIR) image. Diffusion weighted MR image (B) and apparent diffusion coefficient (C) map show increased diffusion in the same region, indicating vasogenic edema

bus or an extrinsic compression, which in turn causes venous pressure increase and eventually progresses to venous infarction. The seriousness of cerebral venous disorders depends on the endogen thrombolysis and/or the existence of collaterals. Patients with CVT often present with cerebral infarcts, wherein retrograde venous pressure and intracranial pressure increases (4). The rising venous pressure associates with the disruption of capillary tight junctions, which in turn increases the volume of extracellular water, leading to the development of vasogenic edema. Subsequently, when the capillary flow becomes significantly reduced, increase in intracellular water will ensue (cytotoxic edema). For this reason, diffusion restriction on diffusion-weighted images and gyral staining due to dysfunction in the blood-brain barrier on postcontrast MRI images may be determined. Gyral or cortical staining commonly associates with venous infarction (5). Isolated CVI is less commonly encountered than dural sinus thrombosis. Its detection depends primarily on visualizing the thrombosed vein and, secondarily, on visualizing any associated venous infarction or haemorrhage. The most sensitive examination technique is MRI in combination with MR venography (6). A variety of parenchymal or cortical changes are seen on MR imaging of venous infarction. These findings vary greatly, depending on whether venous sinuses, superficial cortical veins, or the deep venous system are thrombosed. Generally, parenchymal changes can include edematous and haemorrhagic lesions. On CT and MRI, for patients with cerebral venous thrombosis, cerebral edema due to ischemia does not conform to the pattern of an arterial distribution (7). Venous infarct is rarely non-hemorrhagic. Because the cranial CT is unremarkable; MRI

and MR venography are used for the diagnosis of nonhemorrhagic venous infarction (5). Our three cases had lesions in accordance with venous infarction that do not follow the boundary of arterial territories. One showed restricted diffusion on diffusion-weighted MRI, and two of them showed contrast enhancement on MR images. Isolated CVI without dural sinus thrombosis, as seen in our patients, has rarely been reported. Because of the cortical or superficial involvement of the lesion, we did not find any abnormalities in MRI venography in our three cases. The infarction areas that were determined on MRI did not follow the boundary of classical territories without signs of sinus thrombosis, so a diagnosis of pure cortical venous infarction was made. The spectrum of clinical symptoms depends on the localization of venous infarction. The main presenting symptoms of CVI may be headache, focal neurologic signs, alteration of consciousness, and partial complex or secondary generalized seizures (2, 8). Case 3 presented with secondary generalized tonic-clonic seizures; and case 2 had partial seizures at the follow-up. No focal neurologic deficits were determined in our cases. Anticoagulant therapy with low molecular weighted heparin was administered to three patients and the symptoms recovered completely without any residual sequel. Our patients did not have any hemorrhage. CSF examination was evaluated to rule out encephalitis. However, the CSF examination of our cases was normal. The etiologic mechanisms in pure CVI or dural sinus thrombosis are probably similar. Prothrombotic conditions including pregnancy, oral contraceptive use, infections, and local abnormalities have been found to be potential predisposing factors in CVT. As in our cases, the cause is unknown in approximately 30% of CVT patients (3). Although prothrombotic and autoimmune screens were investigated, the predisposing factor for cortical venous infarction was not found in our patients.

Conclusion

CVI should be suspected in patients who present with sudden onset headache and/or focal epileptic seizures even if there is no focal neurologic deficit. The diagnosis and treatment of CVI should be considered as an emergency because of the high potential for full recovery with anticoagulant treatment.

Conflict of Interest

No conflict of interest was declared by the authors.

References

1. Wasay M, Bakshi R, Bobustuc G, Kojan S, Sheikh Z, Dai A, et al. Cerebral venous thrombosis: Analysis of a multicenter cohort from the United States. *J Stroke Cerebrovasc Dis* 2008;17:49-54. [\[CrossRef\]](#)
2. Karaçayır SS, Balcı K, Asil T, Celik Y. Beyin Damar Hastalığı Sonrası Gelişen Epileptik Nöbetler. *Trakya Univ Tip Fak Derg* 2006;23:127-34.
3. Sagduyu A, Sirin H, Mulayim S, Bademkiran F, Yuntun N, Kitis O, et al. Cerebral cortical and deep venous thrombosis without sinus thrombosis: clinical MRI correlates. *Acta Neurol Scand* 2006;114:254-60. [\[CrossRef\]](#)
4. Schaller B, Graf R. Cerebral venous infarction: the pathophysiological concept. *Cerebrovasc Dis* 2004;18:179-88. [\[CrossRef\]](#)
5. Makkat S, Stadnik T, Peeters E, Osteaux M. Pathogenesis of venous stroke: Evaluation with diffusion- and perfusion-weighted MRI. *J Stroke Cerebrovasc Dis* 2003;12:132-6. [\[CrossRef\]](#)
6. Lai PH, Li JY, Lo YK, Wu MT, Liang HL, Chen CK. A case of spontaneous intracranial hypotension complicated by isolated cortical vein thrombosis and cerebral venous infarction. *Cephalalgia* 2007;27:87-90. [\[CrossRef\]](#)
7. Visrutaratna P, Oranratanachai K, Likasitwattanukul S. Clinics in diagnostic imaging (103). *Singapore Med J* 2005;46:238-44.
8. Benbir G, Ince B, Bozluolcay M. The epidemiology of post-stroke epilepsy according to stroke subtypes. *Acta Neurol Scand* 2006;114:8-12. [\[CrossRef\]](#)